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ANALYSIS OF TWO METHODS OF ISOMETRIC MUSCLE CONTRACTIONS DURING THE ANTI-G STRAINING MANEUVER

by

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1991

A thesis submitted in partial fulfillment
of the requirements for the

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ABSTRACT

Analysis of Two Methods of Isometric Muscle Contractions During the Anti-G Straining Maneuver

by

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This study investigated the difference in Mean Arterial Pressure (MAP) and Cardiac Output (CO) between two methods of isometric muscle contractions during the Anti-G Straining Maneuver (AGSM). 12 subjects (ages 18 to 38 yrs, height 176.8 $\pm$ 7.4 cm, body mass 78.8 $\pm$ 15.6 kg, percent body fat 14.3 $\pm$ 6.6 %) participated in the study. The study was a one-way within-subject design with test conditions counterbalanced. Two methods of isometric muscle contractions lasting 30 seconds each were assessed; an isometric push contraction and an isometric muscle tensing contraction. The dependent parameters were MAP and CO. The average MAP during the push contraction was 123 mmHg, SD $\pm$ 11 and for tense was 118 mmHg, SD $\pm$ 8. CO was 7.6 L/min, SD $\pm$ 1.6 for push and 7.9 L/min, SD $\pm$ 2.0 for tense method. Dependent t-tests revealed $t(11) = 1.517$, $p = 0.157$ for MAP and $t(11) = 0.875$, $p = 0.400$ for CO. This study demonstrated that the two methods of isometric muscle contractions were not statistically different with regards to MAP and CO. Therefore, both forms of isometric contractions may be potentially useful when performing the muscle contraction portion of the AGSM.

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I am grateful for the opportunity afforded to me by the United States Air Force to pursuit my graduate education while serving this great nation.

CHAPTER 1

INTRODUCTION

A century ago, Orville Wright performed aviation's historic milestone of powered controlled flight from Kill Devil Hills, of Kitty Hawk, North Carolina. Since that time technological advances such as rocket and jet powered flight have been introduced. Man has now the ability to fly faster, higher and farther than anyone had thought possible. The early aviation scientists studied the physiological effects of problems such as altitude, space orientation, and weightlessness. There was little need to consider the physiological effects of acceleration and maneuvering at high speed.

Purpose of the Study

The Anti-G Straining Maneuver (AGSM) is a technique used by United States Air Force pilots to attempt to diminish the risk of G induced loss of consciousness, termed G-LOC, by combining a Valsalva maneuver with an isometric co-contraction or tensing of the skeletal muscles. This study is focused on the lower body skeletal muscle straining part of the AGSM. An alternative method of muscle contraction was investigated, which required the subject to push against the floor of the cockpit utilizing maximum isometric extension of the hip and knee. The purpose of the study was to determine whether there was a significant difference in Mean Arterial Pressure and Cardiac Output between the two different methods of muscle contractions.

Research Question

The focus of this study was to determine whether there is a significant difference in Mean Arterial Pressure (MAP) and Cardiac Output (CO) between two different methods of isometric contractions. The research question was: Does Mean Arterial Pressure (MAP) and Cardiac Output (CO) significantly differ between two methods of isometric muscle contractions during the Anti G Straining Maneuver?

Need for the Study

Current United States Air Force physical technique, practiced and performed by aircrew subjected to the effects of centripetal acceleration, incorporate a voluntary isometric tensing contraction of the skeletal muscles and a cyclic Valsalva-breathing maneuver (Burton & Whinnery, 1996). The muscle tensing is performed using all skeletal muscles with special emphasis on the muscles of the lower extremities. Since about 60 percent of the body's blood is found in the venous system, muscle contractions were reported to prevent blood from pooling in the lower body, thereby increasing venous return (Mohrman & Heller, 1997). In addition, this isometric contraction results in an increase in both arterial blood pressure and cardiac output. A Valsalva maneuver is used in conjunction with the isometric muscle contraction to additionally increase arterial blood pressure. The performance of the Valsalva-breathing maneuver consists of trying to forcefully exhale against a closed glottis for 3 to 3.5 seconds. A rapid exhalation and inhalation, during the Valsalva maneuver, taking 1 second or less, allows oxygen to perfuse the lung tissue (Burton & Whinnery, 1996). This cyclic Valsalva technique reportedly results in an increase in G tolerance by contributing to the overall rise in

arterial blood pressure (Burton & Whinnery, 1996). Furthermore, the quick breath results in a decrease in intrathoracic pressure allowing venous return to the heart. The increased arterial pressure achieved through both the isometric tensing of the skeletal muscles and Valsalva maneuver of the current AGSM is critical to counter the physiological effects of extreme G forces (Burton & Whinnery, 1996).

Consideration has been given to the use of an alternative method of isometric muscle contraction of the lower extremities, which requires the subject to push against the floor of the cockpit. This technique is described extensively in the literature and was therefore chosen to be utilized as the alternate form of muscle contraction for the purpose of this study. Despite the prevalence of use in existing studies, little information has been published comparing the physiological responses of the pushing contraction technique to that of the current AGSM tensing muscle contraction. Studies by MacDougall, McKelvie, Moroz, D.E., Moroz, J.S., and Buick (1993), and Kobayashi, Kikukawa, and Onozawa (2002) assume that the tensing maneuver in the currently used AGSM has the same physiological effect as an isometric pushing action.

The purpose of this study was to compare the effectiveness of two different methods of isometric muscle contractions used to increase Cardiac Output (CO) and Mean Arterial Pressure (MAP) during the Anti-G Straining Maneuver (AGSM).

Definition of Terms

1. Body composition – the proportions of fat, muscle, and bone making up the total body, usually expressed as a percent of body fat and percent of lean body mass (Nieman, 1999).

2. Hypertension – a condition in which the blood pressure is elevated above systolic and diastolic measurements of 140 and 90 mmHg, respectively (Nieman, 1999).
3. Isometric muscle contraction – muscle contraction in which the muscle attempts to shorten against an immovable object or when the muscle is voluntarily contracted against its antagonist muscle which results in no change in muscle length (Nieman, 1999).
4. Mean Arterial Pressure (MAP) – the average blood pressure at the root of the aorta (Mohrman & Heller, 1997). MAP represents the average force exerted by the blood against the arterial wall during the entire cardiac cycle. MAP is slightly lower than simply the arithmetic average of the systolic and diastolic pressures, because the heart remains in diastole longer than in systole (McArdle, Katch, F. & Katch, V., 2001). MAP is calculated by the following equation: MAP = 1/3(systolic blood pressure - diastolic blood pressure) + diastolic blood pressure (Nieman, 1999).
5. Valsalva maneuver – a forceful attempt to exhale air from within the lungs against a closed glottis, which increases pressure in the chest cavity (Nieman, 1999).
6. Total Peripheral Resistance (TPR) – the overall resistance to flow throughout the entire systemic circulation (Mohrman & Heller, 1997).
7. Cardiac Output (CO) – the total volume of blood in liters pumped by the heart per minute (Mohrman & Heller, 1997).

Limitations and Assumptions

The limitations of the study were:

1. It was limited to 12 subjects and therefore may not be representative of the general population.
2. It was limited to males.
3. It was limited to testing at 1 G due to the lack of access to a human centrifuge.
4. It was limited to use of non-invasive methods for obtaining Mean Arterial Pressure (MAP) and Cardiac Output (CO) data.

Assumptions:

1. It was assumed that subjects complied with the instructions given to them.
2. It was assumed that subjects performed the methods maximally and according to instruction.
3. It was assumed that the arterial blood pressure was accurately measured.
4. It was assumed that data obtained using bioimpedance cardiography instrumentation was reliable and valid.
5. It was assumed that observation of breathing during testing was sufficient to indicate that the Valsalva maneuver was not used.

CHAPTER 2

RELATED LITERATURE

Before discussing the physiological effects of acceleration and the significance of this study, certain terms should be defined. Most high performance aircraft generate centripetal acceleration that results in centrifugal or inertial type forces (Burton & Whinnery, 1996). Acceleration force (a) and inertial force (G) are expressed in terms of Earth's gravitational constant (g, 9.8 m/sec^2). The ratio a/g, or for inertial forces G/g, has been designated G, a dimensionless quantity evaluating acceleration fields as multiples of Earth gravity, g (Burton & Smith, 1996). The term "G" is also used to define the inertial force resulting from the linear acceleration of gravity acting upon a mass.

When the term G force is used in the flying environment, it is referring to the inertial force resulting from acceleration. As an aircraft accelerates in one direction, inertial forces act on the body in the opposite direction of the applied force. It is the inertial force that gives the pilot the sense of G force. G forces are classified according to the direction of force applied, which can be positive, negative, or transverse forces. If the acceleration force acts in the same direction as normal gravity, from the head toward the feet, the designation is positive G or +G. If the force acts in an opposite direction to that of normal gravity, it is called negative G or -G. Finally, if acceleration forces act perpendicular to normal gravity they are referred to as transverse Gs (Burton & Whinnery, 1996).

Acceleration forces can be further described using the body's relative position to the G force, and are identified using an axial system: longitudinal, lateral, and horizontal (see Figure 1). The three axes are as follows:

1. z axis, is the longitudinal or vertical axis
2. y axis, is the lateral, either right or left axis
3. x axis, is the horizontal axis

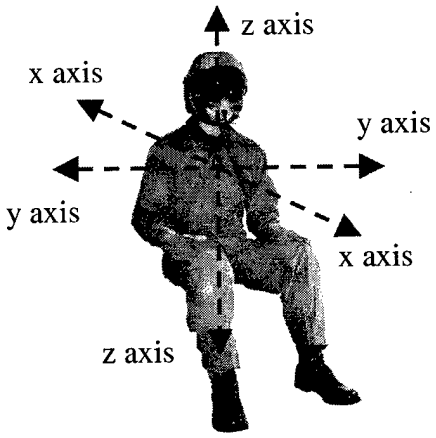

Figure 1

During straight and level flight most acceleration forces are primarily transversal Gs and act in the horizontal direction (G_x). However, during turns, climbs, and dives the force acting upon the body is in the vertical axis (G_z). These maneuvers generate a form of centripetal acceleration, which creates an inertial force that acts upon the body from the head to foot. Although aerobatic flight subjects the body to forces acting in all three axes, the present study focused only on those forces acting in the direction as normal gravity.

The force of positive acceleration ($+G_z$) can be rhetorically described several ways. The initial sensation throughout the body under increased $+G_z$ load is that of a heavy feeling. Arms and legs are difficult to move against the increased "heaviness." Under extremely high G force, soft tissue (i.e. cheeks) will begin to sag. The blood flow to the head tends to decrease and pool in the lower extremities. One of the first manifestations of this blood pooling is that the pilot's vision becomes impaired (Burton

& Whinnery 1996). When $+G_z$ acceleration reaches two (2 G) to four (4 G) times that of normal gravity (1 G), a reduction of peripheral vision resulting in tunnel vision, a loss of color vision, and/or a complete loss of vision (black out) may occur (Burton & Whinnery 1996). These changes are primarily due to an insufficient amount of oxygen supplying the retina caused by a decrease in overall blood supply to the head (Burton & Whinnery 1996; Wood, 1987; Wood, Lambert, Baldes, & Code, 1946). Finally, if the $+G_z$ headward acceleration continues or increases, a state of G induced loss of consciousness or G-LOC may occur. G-LOC is defined as "a state of altered perception wherein one's awareness of reality is absent as a result of sudden, critical reduction of cerebral circulation caused by increased G force" (Burton, 1988). G-LOC may occur without any of the preliminary warning signs of diminished vision loss (Armstrong & Heim, 1938; Burton & Whinnery, 1996).

The exact $+G_z$ magnitude that may causes a particular physiological condition to occur is unique to each individual and is termed G tolerance. A person's G tolerance is determined by the following sequence of events (Burton, 1988; Burton & Whinnery, 1996, 1998):

1. Blood pools in the lower extremities.
2. Since the body is usually in the upright-seated position, the upper body is affected the most due to the fact that the $+G_z$ inertial force is directed from the head towards the feet. The heart must work harder to maintain adequate blood pressure to supply the upper body and brain with blood.
3. Blood vessels constrict in an attempt to prevent the pooling of blood.

Most jet fighter aircraft have a greater thrust to weight ratio, and an increased maneuverability, which means that they can routinely generate and subject the pilot to inertial forces up to 9 G for periods of several minutes (Burton & Smith, 1996). Therefore, to avert G-LOC, a pilot must be able to counteract the detrimental physiologic effects of the imposed G load. A technique used to prevent G-LOC involves straining or tensing the skeletal muscles combined with a Valsalva maneuver. This technique is referred to as the Anti-G Straining Maneuver (AGSM).

There is considerable published literature identifying the problem of G-LOC during flight. One of the first published reports was in 1919 by Dr Henry Head entitled "The Medical Problems of Flying" in which he identified and described a phenomenon called "fainting in the air" (Burton, 1988).

In 1932, the United States Navy identified problems with G-LOC in the Journal of Aviation Medicine. In the article, "Naval Problems in Aviation Medicine", it was suggested that this "fainting in the air," which occurred frequently during dive-bombing missions was attributed to "cerebral anemia produced by centrifugal action" (as cited in Burton, 1988). In the years that followed, considerable research was accomplished identifying and understanding the G-LOC problem. In 1938, P.C. Livingston conducted several comprehensive experiments on G-LOC using a modified bi-wing aircraft that was able to achieve 4 to 6 Gs, causing the test subject, who was usually Livingston himself, to lose consciousness (Burton, 1988). In 1938, Armstrong and Heim carried out the first human studies on the effects of G-LOC involving a centrifuge within the United States (Burton & Whinnery, 1996). The centrifuge used during these early experiments could sustain G loads indefinitely, which provided a unique environment to study the

physiological effects of acceleration and inertial forces. With the use of the centrifuge, research to counteract the physiological effects of acceleration was studied and modified under controlled conditions (Burton, 1988).

During World War II, combat aircraft had advanced in both speed and agility from those used throughout World War I. The Germans were cognizant of the problems of G-LOC with the use of Stuka dive-bombers, which achieved G loads up to 7 G (Burton, 1988). Early German aviation physiologists hypothesized that rapid centripetal acceleration in the $+G_z$ axis diminished blood flow towards the brain, causing G-LOC. Therefore, German pilots were taught to lean forward in an attempt to increase their G tolerance by shortening the vertical distance that their heart would have to pump blood to their brain (Wood et al., 1946; Burton & Whinnery, 1996). This was one of the first documented techniques used as an anti-G countermeasure to prevent G-LOC.

Since an increase in G tolerance cannot be attained by will power alone, a physical technique must be used to increase blood pressure and venous return to counter the effects imposed by an increase in G load. The concept of a muscular straining maneuver to increase G tolerance was first proposed by Steinforth, 1933. It was further investigated by Baldes and Wood at the Mayo Clinic (1943). This straining maneuver, called the M-1, was developed and used in conjunction with an anti-G suit. Anti-G suits consisted of a tightly worn garment that covered the abdomen and legs. Under an increased G load the garment would pneumatically inflate causing and increased peripheral resistance, which aids the muscle tensing efforts. This provided adequate protection for the G loads of their time (WWII). Although a few slight modifications have been made to the M-1 maneuver over the last fifty years, this concept of increased

peripheral resistance through a tensing muscle maneuver is still used as the foundation for achieving G-LOC protection above 5 G (Burton, 1988; Burton & Whinnery, 1996).

Wood and Hallenbeck's research in the 1940s at the Acceleration Laboratory at the Mayo Aero Medical Unit, Rochester, Minnesota, investigated the physiological adjustments to increase G tolerance using the human centrifuge. They were particularly focused on increasing blood pressure (Wood & Hallenbeck, 1946). Using a high-speed centrifuge they experimented with different types of G tolerance enhancement techniques. Much of what is known today regarding anti-G countermeasures and the physiological effects of acceleration was learned from these early experiments (Burton, 1988; Burton & Smith, 1996; Wood, 1992).

The effectiveness of any physical maneuver used to increase G tolerance depends on the ability in which it can enhance arterial blood pressure. This increased blood pressure can maintain cerebral blood flow against inertial forces created by $+G_z$ centripetal acceleration (MacDougall, McKelvie, Moroz, E., Moroz, J.S., & Buick, 1993). The present anti-G straining maneuver (AGSM) was first published and described by Wood and his colleagues in a series of articles published throughout the 1940s. These self-protective maneuvers described by Wood and Hallenbeck (1946) combined a voluntary tensing of the muscles of the arms and legs with a forced Valsalva maneuver against a partially closed glottis.

Additional work by Lambert, Wood and Baldes (1944) as cited in Lohrbauer, Wiley, Shubrooks, & McCally (1972), demonstrated that use of a "weighted control stick" under $+G_z$ centrifugal forces could provide some limited protection from G-LOC. This was accomplished by using a 19-lb. weight and pulley system attached to an aircraft

control stick. This required an increased muscular effort to hold and move the control stick as acceleration increased in the $+G_z$ direction. This application of muscular action against a form of resistance caused an increase in arterial blood pressure. The more centripetal force that was applied to the weighted control stick, the harder it was to move and therefore required an increase in muscle tissue activation and resultant increase in blood pressure.

Lind and co-workers research (1964, 1967) showed that sustained isometric muscular contraction resulted in an increase in arterial blood pressure as long as the contraction was maintained (Lind & McNicol, 1967; Lind, Taylor, Humphreys, Kennelly, & Donald, 1964). This pressor effect of static muscular exercise accounted for G protection as reported by Lambert et al. (1944).

Previous work by MacDougall and associates (1993) compared the effectiveness of several variations of the AGSM for increasing blood pressure. They demonstrated that at 1 G an increase in arterial blood pressure occurred when subjects performed a heavy dynamic weightlifting leg exercise. The increase in blood pressure occurred rapidly when maximal efforts were involved, resulting in elevations of more than 220 mmHg (systolic) and 160 mmHg (diastolic) above that of subject's resting blood pressure (MacDougall et al., 1993). The blood pressure increase with this form of exercise was related to the size of the muscle mass contracted and the intensity of the contraction. This rise in blood pressure was due to the effects of the mechanical compression of blood vessels, known as a pressor response and an elevated intrathoracic pressure caused by a brief involuntary Valsalva maneuver, which accompanied each muscle exertion phase. When subjects performed maximal isometric contractions of the leg muscles, peak

systolic and diastolic pressures were lower than those measured while performing a concentric isotonic contraction at the same intensity (MacDougall et al., 1992). When subjects performed only a maximum Valsalva maneuver (with no lifting), systolic pressure increased approximately 135 mmHg and diastolic approximately 90 mmHg over resting blood pressure. When forceful dynamic leg contractions only were performed at intensities that did not require a Valsalva maneuver, an overall increase in blood pressure was reported to have occurred (MacDougall et al., 1992).

Studies by MacDougall et al. (1993) and Kobavashi et al. (2002) involving the application of the AGSM refer to the muscle contraction action as a pushing motion. Although, a pushing motion is not the method that is presently taught by the USAF, it does allow for a means by which to measure the force generated by the muscles while performing the contractions. Unfortunately, this is not the method either used or practiced by aircrew members to counteract the physiological effects of G forces. The correct method of muscle contraction used during the AGSM consists of an isometric tensing contraction of all the skeletal muscles with special emphasis on the lower extremities of the abdomen, buttocks, and leg regions (Burton & Whinnery, 1996).

The increase in blood pressure while performing the AGSM is the combined effect of two separate mechanisms, a forceful contraction of the skeletal muscles and intrathoracic pressured created by the Valsalva maneuver. The maneuvers are independent of each other and contribute to maximize an increase in blood pressure (MacDougall et al., 1993).

When muscle contractions are sustained, the blood pressure rises in proportion to the tension or effort exerted by the muscles. During the muscle tensing of the isometric

contractions, blood pressure increases and remains constant throughout the duration of tension as compared to that of dynamic contractions in which blood pressure increases and decreases throughout the full motion of muscle shortening (Lind et al., 1964).

The AGSM technique currently used by U.S. pilots under high G environments utilizes a forced exhalation against a closed glottis while tensing the muscles of the legs, arms, and abdominal muscles (Burton et al., 1996). This technique increases the intrathoracic pressure up to 100 mmHg. To allow for adequate venous return to the heart, the intrathoracic pressure produced from this Valsalva maneuver must be interrupted every 3 to 3.5 seconds with a rapid expiration/inspiration process. This respiratory cycle lasts no more than 1 second. This brief period allows both adequate venous return and ventilation when the intrathoracic pressure is diminished (Burton & Whinnery, 1996).

The increased systemic arterial pressure generated by the Valsalva maneuver is well documented (Balldin, 1983; Burton, Leverett, & Michaelson, 1974; Burton & Whinnery, 1996; MacDougall et al., 1993; Shubrooks & Leverett, 1973) and was not examined in this study.

The cardiovascular effects associated with static exercise are referred to as the "pressor response" (Bryant & Peterson, 1998). This term refers to the effect of alterations in heart rate and blood pressure, which occur reflexively via feedback from the contraction of muscles involved during the static exercise. Three main factors control the intensity of the pressor response. These include a centrally mediated stimulation of the cardiovascular system referred to as the "central command, mechanical vascular compression by the contracting muscle, and vasoconstriction in the non-contracting muscles" (Porcari & Curtis, 1996).

The term "central command" is used to refer to input from the brain to the heart and peripheral vascular system during muscle contraction. This concept suggests that the same cortical drives that initiate voluntary skeletal muscle activity are also responsible for cardiovascular and respiratory adjustments that are essential for the performance of the muscle activity (Mohram & Heller, 1997). The "central command" stimulates the cardiovascular center in the brain proportional to the magnitude of the skeletal muscle activity being performed. As the brain sends impulses to initiate muscle contraction, a proportional number of impulses are also sent to the cardiovascular center in the brain. These impulses increase heart rate and blood pressure supporting muscle activity occurring throughout the working muscle. The resulting activation of the cardiovascular system is proportional to the percentage of maximal strength at which the muscle contracts (Porcari & Curtis, 1996).

The second factor responsible for the pressor response relates to the degree of intramuscular vascular compression as the muscles contract. In both dynamic and static muscle contraction, muscle fibers exert a mechanical compression on the blood vessels leading into and out of the working muscles. This creates an increase in vascular resistance for blood flowing to and from the working muscles (Mohram & Heller, 1997; Porcari & Curtis, 1996; Lind et al., 1964). Total Peripheral Resistance is an indicator of an increase in muscle restricted blood flow caused by changes in muscle contraction. A discussion of the effects of muscle contractions would not be complete without exploring the relationship between Mean Arterial Pressure (MAP), Cardiac Output (CO), and Total Peripheral Resistance (TPR) (TPR = MAP/CO). As more muscle mass is contracted,

there is greater constriction of blood flow, causing an increase in TPR (Guyton, Jones, & Coleman, 1973; Smith & Kampine, 1990).

The final component in the pressor response is the vasoconstriction within the non-contracting muscles. While blood vessels in the contracting muscle are dilated in an attempt to provide more blood and remove waste products, vessels within the inactive muscles are constricted. Sympathetic stimulation causes vasoconstriction in an attempt to shunt blood to where it is needed most (Porcari & Curtis, 1996). The magnitude of the effect of this mechanism can be realized by examining resulting changes in cardiac pressures related to increased venous return to the heart. While at rest, the normal input pressure of the blood to the heart is about 6 to 8 mmHg. For every additional 1 mmHg increase to the input pressure, the output pressure (systolic blood pressure) of the heart is increased approximately 4 to 6 mmHg (USAF Aerospace Physiology Study Guide Workbook, 1993).

The pressor response in exercise is well documented (Franke, Boettger, & McLean, 2000; Miles, Li, Rinard, Clarkson, & Williamson, 1997; Williams & Lind, 1987) and is believed to be controlled by a balance of all three mechanisms working together. Although, the current literature regarding these mechanisms is conflicting, with many articles suggesting that the central command component plays a greater role, while others have reported that peripheral components play a greater role in producing the cardiovascular changes (Friedman et al., 1992, MacDougall et al., 1992, Williams & Lind, 1987). It is generally agreed upon, however, that by improving the quality and intensity of the muscle tensing, both the heart's output blood pressure (mean arterial

pressure) and cardiac output are improved and G tolerance is therefore increased (USAF Aerospace Physiology Study Guide Workbook, 1993).

Isometric exercise is associated with an increase in heart rate (HR) and blood pressure (BP). Smolander et al. (1997) found HR and BP response to isometric exercise in older vs. younger men, increase during isometric muscle contraction and is proportional to the intensity of the contraction effort. Increasing HR proportional to increased levels of force and muscle group size suggests an influence of a muscle-brain reflex that is affected by the number of motor units involved in the contraction. Studies have focused on identifying differences in the pressor effect by varying the muscle groups used in the exercise (hand grip vs. leg extension) and found no significant difference. This suggests that the central command component may be more important than the localized muscle action in the elevation in blood pressure generated during isometric activity (MacDougall et al., 1992, Williams & Lind, 1987). Peripheral response was believed to provide feedback from the contracting muscle to the brain, which in turn increases arterial pressure. This does not appear to be fully understood, as the same study suggested that it was the relative effort of the isometric muscle contraction that was responsible for the increase in arterial pressure achieved with the two differing muscle actions in spite of a large deficit in overall force produced. That is, the handgrip generated far less overall force than did leg extensions yet they yielded same pressor effects (MacDougall et al., 1992).

Cardiac output is the product of heart rate and stroke volume. Since stroke volume is related to venous return, any increase in pressure within the venous system, under increased G significantly reduces venous return. With a reduction in stroke

volume, heart rate increases to maintain adequate cardiac output. As G levels increase, even with an increased heart rate, cardiac output decreases due to reduced stroke volume. Therefore increasing venous return is critical to physiologically enable increased G tolerance. Hence, the straining maneuver employs the use of muscular tensing throughout the skeletal muscles especially the lower trunk and legs to assist with venous return. In return, cardiac output is maintained at an adequate level to support arterial pressure and blood flow to the head and brain (Burton & Whinnery, 1996).

CHAPTER 3

METHODOLOGY

Subjects

This study was approved by the University of Nevada, Las Vegas, Human Subjects Review Board (see Appendix B3). Twelve volunteers were recruited to participate in this study. Males from the University of Nevada were used as subjects for the study. Subjects were between 18 and 38 years of age and were screened for hypertension. Table 1 presents the physical characteristics for each of the 12 test subjects in this study.

Table 1: Physical Characteristics of Test Subjects

Subject	Age	Weight (Kg)	Height (cm)	% Body Fat (Sum of 4 Skinfolds)
1	27	87.0	183.0	15.1
2	25	72.5	175.0	11.6
3	22	72.0	173.5	18.6
4	23	81.0	177.0	5.2
5	24	72.7	174.0	15.1
6	24	106.7	192.5	24.2
7	38	74.5	177.0	18.6
8	22	106.0	182.0	23.5
9	22	77.0	179.5	13.1
10	19	82.0	176.0	15.4
11	24	60.0	162.5	6.5
12	18	54.5	169.5	4.4
Average	24	78.8	176.8	14.3
SD	5.0	15.6	7.4	6.6

Volunteers with a resting blood pressure of $\geq$ 140/90 mmHg (Nieman, 1999) were eliminated from the subject pool. Throughout the testing process only one student was unable to continue with the protocol due to hypertension.

The principle investigator was a United States Air Force Aerospace Physiologist qualified to provide instruction on the performance of the Anti-G Straining Maneuver (AGSM). Males only were used to simplify the statistical treatment.

This study examined both Mean Arterial Pressure (MAP) and Cardiac Output (CO) under two different experimental methods of isometric muscle contractions. The first method consisted of the isometric muscle tensing currently used by the United States Air Force, called the Anti-G Straining Maneuver. Although the performance of the AGSM encompasses the majority of the body's skeletal muscles, the focus of this study was on the lower extremity muscles only. The second method consisted of an isometric muscle contraction resulting from pushing against an immovable surface. This surface was configured with a load cell which measured the strength exerted and displayed it on a monitor providing immediate feedback to the test subject.

During both methods, isometric muscle contractions were performed without the aid of a Valsalva maneuver. Breathing was strictly monitored to guarantee that the Valsalva maneuver was not involved. The digital display, indicating the force of the push, allowed the subject to match his effort during and throughout the three pushing trials.

Thoracic bioimpedance cardiography and arterial blood pressure data were collected during both methods of muscle contractions.

Method/Design

This study was a one-way within-subject design with test conditions counterbalanced. Two methods of muscle contraction lasting 30 seconds each were assessed. The first method consisted of an isometric muscle tensing contraction of the lower extremities. This contraction used an isometric tensing maneuver in which all the muscles of the buttocks and legs were simultaneously contracted. The second condition consisted of a maximal, isometric pushing movement against an immovable footrest platform configured with a load cell, which measured the force of the push and also provided feedback to the participant. The dependent parameters were Mean Arterial Pressure (MAP) and Cardiac Output (CO). Data were collected during three consecutive testing trials for maximum peak contraction lasting 30 seconds each. Subjects were in a seated position in a simulated cockpit throughout the testing conditions. The subject was secured in a seat by a five-point harness, which immobilized the upper body. The legs were resting on a footrest platform equipped with a load cell configured to measure the force produced by leg and knee extension.

Procedures

Arterial Blood Pressure Measurement

Arterial blood pressure (systolic/diastolic) was measured from the left upper arm so it constricted the brachial artery at heart level according to the American Heart Association procedures. A systolic and 5th phase diastolic blood pressure was taken during three separate trials for each of the two methods of muscle contractions using a manual mercury blood pressure sphygmomanometer. The blood pressure measurements were

taken once within each of the 30-second trials for the two different isometric muscle contraction methods.

Maximum Isometric Push

After a familiarization session with the test equipment, maximum voluntary strength produced by the lower extremity muscles, primarily the hip flexors and knee extensors, during the push condition was determined using an electronic load cell[1] attached to a footrest platform. Force produced by the pushing motion was measured to monitor and reproduce the intensity of the push contraction being evaluated (see Appendix A2). The test subject was in a seated position held in place by a five-point harness, which immobilized the upper torso and provided a means of static resistance against which the subject forcefully pushed. Breathing was monitored to prevent the use of a Valsalva maneuver throughout the push contractions.

Isometric Muscle Tensing

Isometric tensing of the lower extremities involving both extensor and flexion muscles were performed with the subject seated and restrained by a five-point harness. Breathing was strictly monitored to prevent the use of a Valsalva maneuver. Subject's feet were positioned on a footrest plateform, which was configured with an electronic load cell allowing for immediate feedback to prevent the pushing contraction method from being used. Isometric tensing contractions lasted 30 seconds each.

Body Composition Measurements

Determination of height, weight, and percent body fat was part of the initial orientation session. Four skinfold measurements using a Lange[2] skinfold caliper and the

[1] Transducers Inc., model T363-500-20P1 (Whittier, California)
[2] Beta Technology Incorporated (Cambridge, Maryland)

Jackson and Pollock sum of four skinfolds equation determined percent body fat. This equation is reliable for both men and women and uses age as a factor within the equation. It correlates 0.94 with body composition determined by underwater weighing techniques (Golding, 2000).

The four skinfold sites used were (Golding, 2000):

a. Abdomen-vertical fold - 1 inch to the right of the umbilicus
b. Illium-diagonal fold - just above the crest of the illium on the midaxillary line
c. Thigh-vertical fold - midway between the top of the patella and groin line
d. Triceps-vertical fold - measured on the back of the upper arm midway between the acromion and olecranon processes

The sum of the four measurements ($\sum_4$) was entered into the Jackson and Pollock equation based on age and gender to determine percent body fat.

The Jackson and Pollock sum of four skinfolds equation for men is (Golding, 2000):

$$\text{Percent Fat} = 0.29288\ (\textstyle\sum_4) - 0.005\ (\textstyle\sum_4)^2\ 0.15845\ (\text{Age}) - 5.76377$$

The standard error (SE) for the men's equation is 3.49% fat and R = 0.901 (Golding, 2000).

The Lange Caliper meets the specifications established by the committee of the Food and Nutrition Board of the National Research Council of the United States (Golding, 2000). They have a jaw surface area of 30 mm^2. In addition, the Lange Calipers have a standard jaw pressure of 10g/mm^2 providing a constant jaw spring tension pressure of 300g regardless of the width of the calipers (Golding, 2000).

Bioelectrical Impedance Cardiography

Stroke volume and heart rate were continuously measured throughout the thirty-second muscle contractions for both methods. An Ambulatory Impedance Monitor[3] (AIM-8) cardiography unit was programmed to measure a 20-second average across the 30-second testing time period. Correlation coefficients between impedance cardiography and traditional invasive methods of cardiac output were reported to yield correlation coefficients of 0.7 to 0.8 (Buell, 1988). Published validity studies also compared heart rate, pre-ejection period, left ventricular ejection time, and stroke volume between the Ambulatory Impedance Monitor (AIM-8) and the commonly used Minnesota model 304B impedance cardiography monitor. All Pearson R correlations were > +0.87, and all p were < 0.001 (Sherwood, McFetridge, & Hutcheson, 1998).

The AIM-8 impedance cardiograph supplies a constant 2 mA sine-wave current to impedance electrodes #1 and #4 at a frequency of 80 kHz, and detects the resulting voltage developed due to the bioelectric impedance at electrodes #2 and #3 (see Figure 2). An electrocardiogram (ECG) signal is detected at electrodes #5 (ECG+) and #1 (ECG-). The AIM-8 uses these signals to compute the various cardiac performance indices. A manual start button located on the outside of the AIM-8 allows activation of the unit to initiate recording of measurements.

Leads #1 and #4 are used as the current excitation electrodes, and are terminated with spot electrode clips. Lead #5 is used as a positive ECG spot electrode. The ECG signal is detected between electrodes #1 and #5, and is referenced to electrode #4. The 5th electrode allows the user to easily move the electrode around the subject's left side to

[3] Bio-Impedance Technology, Inc., (Chapel Hill, North Carolina)

achieve the best ECG signal. Leads #2 and #3 are the voltage detection leads and are used with the Mylar band electrodes (#2 and #3) (AIM User's Reference Manual, 2002).

The sensor electrodes and leads were placed on the body according to the manufacturer's specifications (Figure 2.). The recommended electrode arrangement was specifically developed for use with the AIM-8 monitor, consisting of a tetrapolar combination of spot and band electrodes. The voltage recording electrodes are the two Mylar band electrodes placed around the base of the neck and the thorax, which crosses the tip of the xiphoid process. The AIM-8 electrode lead wire #2 connects to the band electrode located at the base of the neck, and lead wire #3 connects to the band electrode located around the base of the thorax at the tip of the xiphoid process. The lead wires are attached to the Mylar band electrodes by folding each of the two ends of each Mylar bands back away from the two adhesive surfaces and clipped, using alligator clips, to the center metal portion of the bands. Disposable ECG spot electrodes are used as the current electrodes. A spot electrode and lead wire #1 is placed behind the right ear over the base of the mastoid process. The other electrode and lead wire #4 is positioned over the lower right rib cage, 6 cm below the lower recording Mylar band electrode (#3). The positive ECG spot electrode and lead wire #5 should be placed on the lower left rib cage. Electrode #5 position may be adjusted as needed for best ECG signal. The two current electrodes (#1 and #4) along with the 5th lead (ECG+) and electrode serve as a source for the ECG signal to the AIM-8 monitor similar to a lead II ECG configuration (AIM User's Reference Manual, 2002).

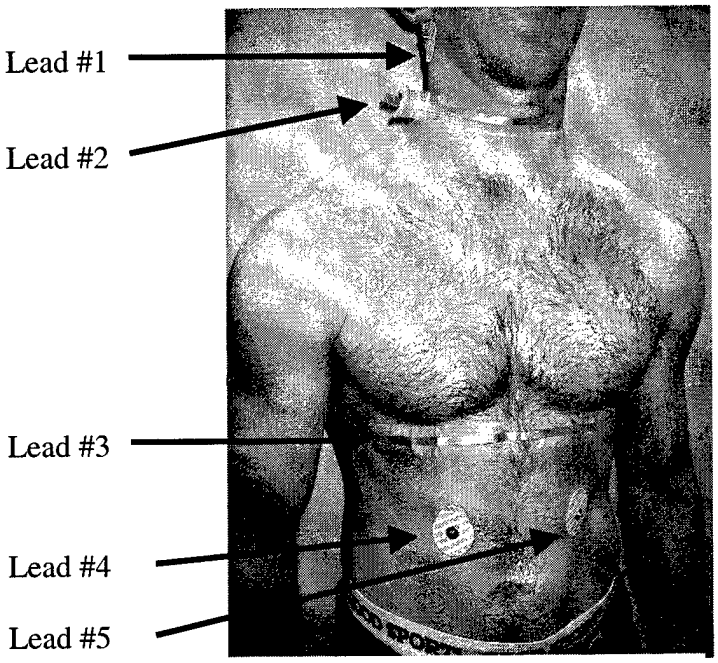

Figure 2

Lead #1	Electrode - behind right ear at base of mastoid process
Lead #2	Band - surrounding the base of the neck
Lead #3	Band - surrounding lower thorax crossing tip of xiphoid process
Lead #4	Electrode - lower right rib cage 6 cm below the #3 electrode
Lead #5	ECG Electrode - lower left rib cage 6 cm below the #3 electrode

Bioimpedance data were converted to usable information using COPWORKS 5.0[4] (Cardiac Output Program Workstation for Windows) editing and analysis software.

Impedance cardiography technology was introduced in the early 1930s, but was later refined for use by the National Aeronautics and Space Administration (NASA) for use as a noninvasive hemodynamic monitoring system for the Apollo space program in the 1960s (Buell, 1988).

[4] Copworks, Bio-Impedance Technology, Inc., (Chapel Hill, North Carolina)

The principle theory of this technique uses resistance to an alternating current, which is known as electrical impedance, to measure electrical resistance or changes that occur within the thorax. If the electrical current remains constant, this resistance or impedance is inversely proportional to voltage. The properties of a conductor are related to the resistance of the conducting medium, the length of the conduit, and its mean cross-sectional area. The AIM-8 thoracic bioimpedance analyzer provides a low-energy, high frequency, alternating electrical current through the thorax to be measured. Electrodes and Leads #3 and #4, located inside the current path, detect the electrical impedance changes. The frequency of the delivered current is very low (80 kHz) and cannot be sensed by the test subject (Buell, 1988). Impedance (Z) changes are generated from blood volume and flow velocity, which increases and decreases in the ascending aorta during systole and diastole. Figure 3 from the Thoracic Electrical Bioimpedance Technology Web Site (http://www.hemosapiens.com/teb.html) represents the timing relationship between ECG, delta Z and dZ/dt signals. The cardiac cycle starts with a contraction at the Q-time of the ECG QRS complex. The Pre-Ejection Period (PEP) is defined as the elapsed time between the Q-time of the QRS complex and the opening of the aortic valve. The ejection phase, which is outlined by the Left Ventricular Ejection Time (LVET or VET), starts with the opening of the aortic valve and ends with the closing of the aortic valve (S2-time). During the initial part of ejection phase the aorta is distended which causes the thorax to becomes more conductive due to the increase in blood volume. The rate of cardiovascular impedance, which changes over time (dZ/dt), is designated as the first derivative of Z, and is a representation of blood flow through the aorta. The maximum value, (dZ/dt)max, is proportional to the aortic blood peak flow.

Impedance to electrical current decreases (increased conductance) during systole due to an increased blood volume, and flow velocity. This impedance changes directly reflect aortic blood flow and left ventricular function.

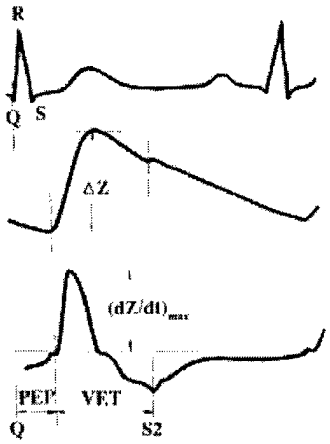

Figure 3

The base thoracic impedance (Zo), pulse impedance/time changes (dZ/dt), along with ECG data are used to calculate stroke volume, cardiac output, and contractile properties of cardiac function. Once the data is collected the COPWORKS software calculates stroke volume based on the Kubicek equation.

Kubicek equation:

$$\text{Stroke Volume (SV)} = \frac{(\rho)(\text{LVET})((dZ/dt)\text{max})(L)^2}{(Zo)}$$

ρ = 135 ohm cm (constant blood resistivity)
LVET = left ventricular ejection time in milliseconds
(dZ/dt)max = maximum value of the first derivative of thoracic impedance in ohms per second
L = mean distance between the two inner band electrodes (#2 and #3) in centimeters
Zo = mean body impedance between electrodes #2 and #3 in ohms

Electromyography (EMG)

Electromyography tracings from the skin surface of several large muscles of the lower extremities were assessed. Muscle sites were identified and prepared prior to electrode placement. The test subject's skin was abraded and cleaned with alcohol prior to surface electrode placement. The following muscles were identified for electrode placement:

1. Gluteus Maximus
2. Rectus Femoris
3. Vastus Medialis
4. Vastus Lateralis
5. Biceps Femoris
6. Gastocnemius Medial

A seventh electrode was used as a ground and was placed on the bony process of the head of the Fibula. All electrodes were affixed to the belly of the above-identified muscles. EMG muscle activation data was collected post data collection on three random subjects to assess if the subjects were adhering to muscle contraction instructions (Appendix C13). Results were used for discussion purpose only and were not used as a dependent measure. Electromyography measurements were recorded using the Noraxon Myosystem 2000.

Experimental Protocol

Test Session

Prior to the collection of data, subjects were afforded an orientation session consisting of an overview of the study and familiarization with the instrumentation and equipment. All subjects were asked to adhere to all instructions.

Subjects read and signed an informed consent document (see Appendix B1), and any and all questions regarding the study were answered to their satisfaction. Resting blood pressure was determined from the left arm. Any subject with a blood pressure measurement greater than 140/90 was eliminated from the study. Without shoes, standing height in centimeters and weight in kilograms were measured. In addition, body composition was estimated using the Jackson and Pollock sum of four skinfolds equation. Skin sites for placement of the bioimpedance cardiography electrodes were identified and prepared by shaving any excess hair and cleaning with an alcohol swab.

The subjects then proceeded to the experimental portion of the study. Both isometric muscle contractions were performed while strapped to a simulated cockpit seat using a five-point harness. An isometric muscle contraction of the lower extremities was performed while pushing against an immovable footrest platform (activation of primarily the hip flexors and knee extensors). A series of three push contractions lasting 30 seconds each were assessed. The second method of isometric muscle contraction was a form of maximum leg muscle tensing of the lower extremities (activation of both extensor and flexion muscles). Three trials lasting 30 seconds each were measured.

The purpose of the harness was to stabilize the body and provide a form of resistance while pushing against the footrest platform and while initiating the isometric

tensing muscle contractions. Bioimpedance data and arterial blood pressure were measured throughout both muscle contraction methods. Thoracic impedance measurements were initiated immediately after the subject started contracting his lower extremity muscles for a total of 20 seconds within the 30 second contraction testing window. At the same point, a manual blood pressure measurement was also initiated within the thirty-second contraction. Once successful collection of data was indicated, the subject was allowed to relax for several minutes before the next contraction condition.

Contraction Methods

1. Isometric muscle contractions of agonist/antagonist lower extremities (maximum leg muscle tensing used during the AGSM; activation of both extensor and flexion muscles).
2. Isometric muscle contraction of lower extremities while pushing against an immovable footrest platform (activation of primarily the hip flexors and knee extensors).

Test Conditions

1. The first method was the current Air Force AGSM consisting of an isometric muscle tensing contraction of the lower extremities (no Valsalva).
2. The second method was an isometric muscle contraction of lower extremities while pushing against immovable force plate (no Valsalva).
3. Three consecutive testing trials, lasting 30 seconds, were conducted for each of the isometric contraction methods.
4. Subjects were in a seated position throughout all testing conditions.

Variables Monitored

1. Cardiac Output (CO) – Cardiac Output was estimated using cardiography impedance technology (Ambulatory Impedance Monitor: AIM-8) throughout the thirty-second muscle contractions trials. CO was calculated from the indirect measurement of Stroke Volume (SV) and Heart Rate (HR) determined from the impedance cardiography unit (HR x SV = CO)(Appendix C3).
2. Stroke Volume (SV) – Stroke Volume was determined from impedance cardiography during both muscle contraction methods using an Ambulatory Impedance Cardiography unit (AIM-8)(Appendix C5).
3. Heart Rate (HR) – Heart Rate was continuously measured by an ECG in conjunction with the bioimpedance analyzer throughout the thirty-second testing contractions (Appendix C7).
4. Force of push contraction – A Transducers Inc. load cell and monitor were used to measure the amount of force produced when pushing with the legs against the footrest platform of the simulated cockpit. The strength in kilograms was displayed on a monitor to provide constant feedback during testing. Indicated strength in kilograms was later converted to pounds and then to force in Newtons (Appendix A2).
5. Mean Arterial Pressure (MAP) and Total Peripheral Resistance (TPR) were calculated from the following equations (Appendix C7, C9):
    a. MAP = Diastolic Pressure + 1/3(Systolic Pressure – Diastolic Pressure)
    b. TPR = MAP/Cardiac Output

Data Analysis

The independent variables were two methods muscle contractions consisting of an isometric push contraction and an isometric tense contraction. The dependent variables were Mean Arterial Pressure (MAP) and Cardiac Output (CO). Both MAP and CO were calculated using an average of the three trials for comparison between the two contraction methods. Each trial of muscle contraction was performed for a thirty second duration or until all data was successfully collected.

This study used a two-tailed dependent t-test to compare both MAP and CO values between two experimental conditions. Microsoft Excel[5] statistical software was used to analyze the data collected. Statistical significance was accepted at $p \leq 0.05$. The study was a one-way within-subject design with test conditions counterbalanced.

[5] Microsoft Corporation (Redmond, WA)

CHAPTER 4

RESULTS

For simplicity the present USAF Anti-G Straining Maneuver (AGSM) is referred to in the figures as "Tense" and the pushing against the footrest platform as "Push." Average Mean Arterial Pressure (MAP) for both muscle contractions was not significantly different between the two experimental conditions ($t(11) = 1.517$, $p = 0.157$). Figure 3 shows the average MAP during the push isometric muscle contraction method was 123 mmHg (SD $\pm$ 11) and 118 mmHg (SD $\pm$ 8) for the isometric tensing method.

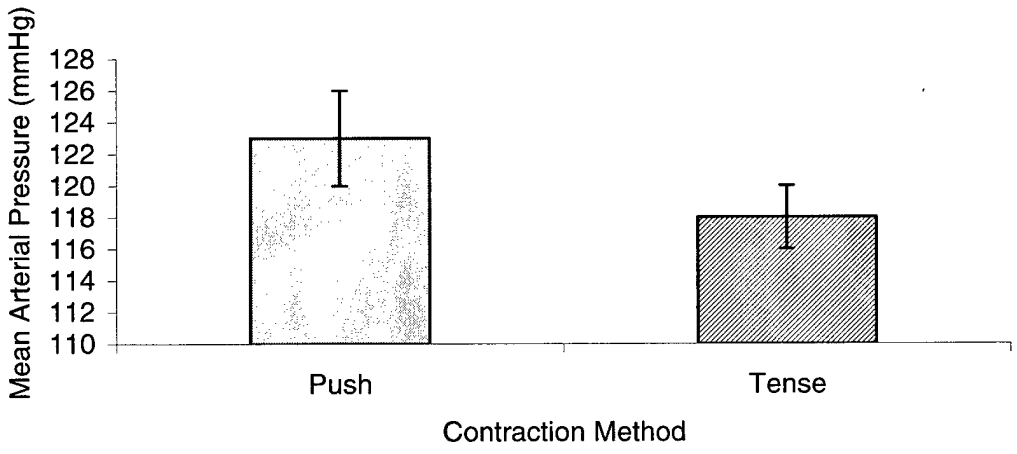

Figure 3: Mean Arterial Pressure vs. Two Methods of Isometric Muscle Contractions. Vertical bars indicate $\pm$ SD (n = 12).

Cardiac Output (CO) was not significantly different between the push and tense experimental conditions ($t(11) = -0.587$, $p = 0.569$). Figure 4 represents the average CO measured while performing the isometric push contraction was 7.6 L/min, SD $\pm$ 1.6. The tense contraction measured an average of 7.9 L/min, SD $\pm$ 2.0.

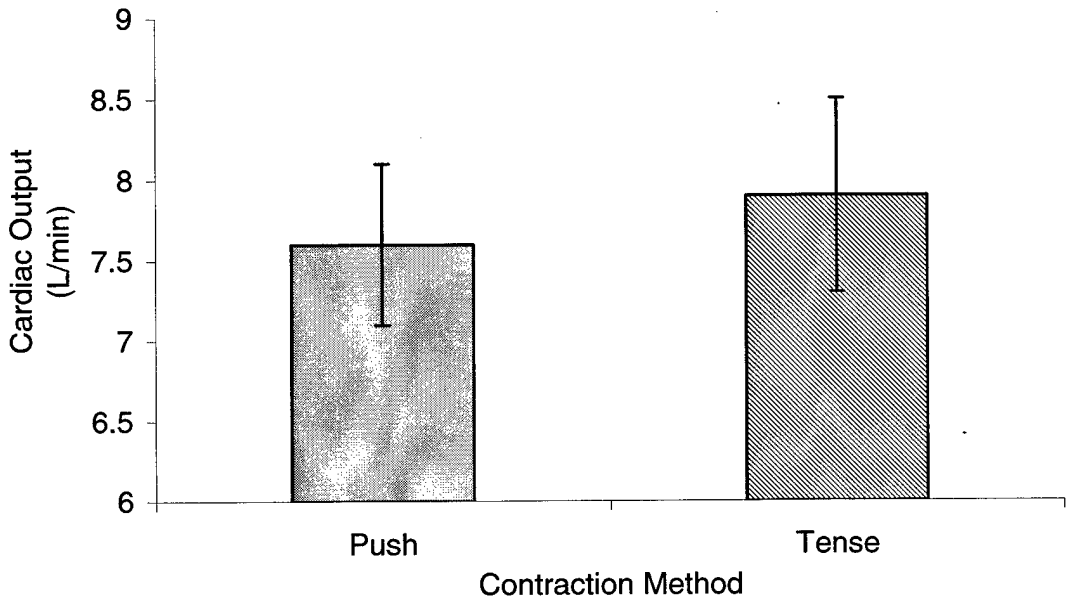

Figure 4: Cardiac Output vs. Two Methods of Isometric Muscle Contractions. Vertical bars indicate $\pm$ SD (n = 12).

Total Peripheral Resistance (TPR) was not significantly different between both muscle contraction conditions ($t(11) = 0.875$, $p = 0.400$). TPR during the isometric Push contraction was 16.6 mmHg/L/min, SD $\pm$ 3.2, compared to Tense contraction which was 15.8 mmHg/L/min, SD $\pm$ 4.3. Figure 5 illustrates TPR for both conditions.

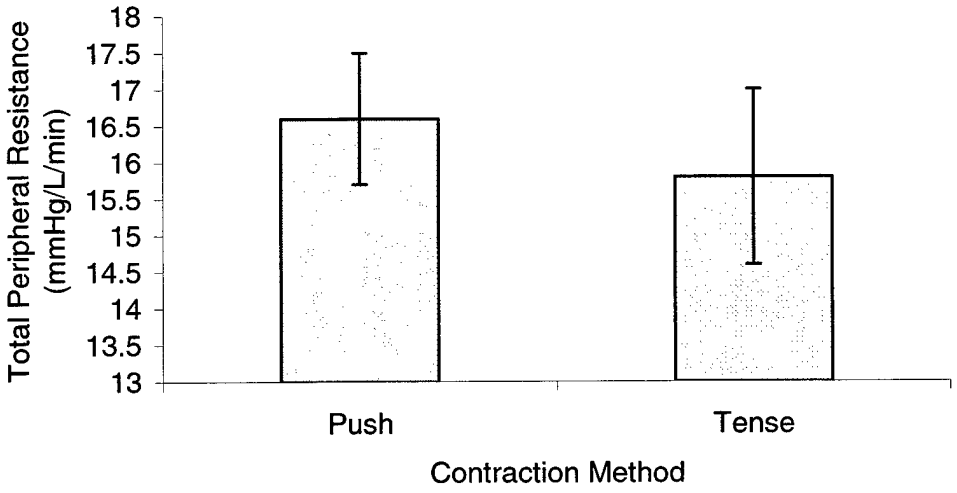

Figure 5: Total Peripheral Resistance vs. Two Methods of Isometric Muscle Contractions. Vertical bars indicate $\pm$ SD (n = 12).

CHAPTER 5

DISCUSSION AND CONCLUSION

Discussion

The focus of this study was to determine whether there were significant differences in Mean Arterial Pressure (MAP) and Cardiac Output (CO) between two different methods of isometric muscle contractions used in the performance of the Anti-G Straining Maneuver (AGSM). The results of this study demonstrated no significant difference between the two conditions of push and tense isometric muscle contractions. This chapter explores possible explanations for the obtained results.

A review of current literature demonstrated a lack of studies that directly compared the two methods of muscle contractions. The "push" method of isometric contraction seems to be favored in the literature for use in a majority of studies, in part, due to the ability to measure the magnitude of effort using a simple load cell (MacDougall et al., 1993, and Kobayashi et al., 2002). Similar methods of measurement for the "tense" method have not been described. As per research by MacDougall et al. (1992), the results of this study support existing data showing that isometric contractions of the same relative intensity elicit a pressor response of the same magnitude despite differences in absolute force generated.

Assurances were made that all study subjects received the same, detailed instruction for performing the two types of muscle contractions. Subjects were instructed

to give their maximal effort for each test trial, and it was felt that there was no bias in performance for either method. However, feedback to the subject regarding their maximal effort could only be provided for the push contraction method.

Post hoc studies were conducted to investigate the possibility of improper performance of the muscle contractions. Electromyography was used to evaluate the effectiveness of instructions for both methods of isometric contraction (see Appendix C13 and C14). Three subjects were randomly selected post data collection, to ascertain compliance with muscle contraction instructions. Six muscles (Gluteus Maximus, Rectus Femoris, Vastus Medialis, Vastus Lateralis, Biceps Femoris, Gastrocnemius Medial) were monitored. It was observed that two of the three subjects had expected results, indicating predominance of contraction of the leg extensors during the pushing isometric contraction method, and contraction of all of the monitored musculature during the tensing isometric contraction method. This demonstrates the likelihood that the subjects were performing the technique properly. Consideration should therefore be given for the use of electromyography to confirm the subject's compliance with instructions in any future testing.

Avoidance of the use of the Valsalva maneuver during maximal muscle contraction was an important feature in the test protocol. Each subject was instructed to breathe normally and refrain from performing the Valsalva maneuver. Direct observation to monitor normal breathing of the subject was used according to a similar study by Misner et al. (1990). Continuous spirometry could be utilized to more reliably monitor the subject's compliance with the breathing instructions.

Additional consideration must also be given to the experience of the subject population with performance of the AGSM. If the study were repeated on pilots who have practical experience in the increased G environment and use of this method, the results may be very different. However, there may also be some bias in performance of the maneuver favoring the tense contraction since this is what the USAF routinely instructs.

The instrumentation used to measure arterial blood pressure and cardiac output data was assumed to be reliable and accurate as noted in Chapter 3. An inherent inaccuracy in instrumentation or measurement methods would presumably have affected the results between the contraction methods to a relatively equal degree. While unforeseen errors might have yielded inaccurate results, overall error would likely have been constant or uniform. Such error would not have favored either muscle contraction method, and the relationship between the results preserved.

The timing of the blood pressure and cardiac output measurements may have also affected the results. Non-invasive monitoring techniques were utilized in this study for the measurement of blood pressure and cardiac output. A manual blood pressure measurement was obtained at a point in time within the 30-second muscle contraction window. The AIM-8 bioimpedance analyzer collected data for an average of 20 seconds within the contraction time frame of 30 seconds. This was the minimum collection time recommended by the manufacturer. The data obtained is supported by research by Lind and associates (1964), which demonstrated a constant elevation in blood pressure during maintained isometric muscular contractions, versus that seen with dynamic muscular

contractions. Use of invasive monitoring devices could provide a more precise data collection but would require considerable changes to the study design.

Random errors in measurement, such as an inaccurate blood pressure measurements or invalid cardiac output readings were addressed throughout the study. Subjects were asked to perform three independent trials for each contraction method. If there was an immediately recognized error or failure in data collection within the bioimpedance analyzer, an additional trial was conducted to acquire the necessary data. Only trials that yielded no measurable data were retested. Errors in reliability of the results could be further reduced by increasing the number of subjects and or the number of trials performed for each contraction method.

Perhaps the greatest limitation of this study was that it was conducted at 1 G. The purpose of this study, with the regards to the performance of the AGSM, was to increase G tolerance by facilitating a muscle contraction maneuver. Reproducing this study using a human centrifuge to produce an increased G environment would yield more valuable results. It is unclear if the data and conclusions gathered at 1 G could be utilized to predict results at multiple G loads. Direct comparison of the methods in an increased G environment may yield entirely different results from the current study. It is noted that it would be difficult to isolate the effects attributable to lower extremity contractions alone since large increases in G require the use of the Valsalva to successfully counteract the forces causing G-LOC. Performing this study at relatively low G load level to test the lower extremity contraction methods alone should be considered for future studies.

Another approach to study at an increased G load, is to consider the performance of the full AGSM maneuver including the Valsalva maneuver, and compare both muscle

contraction methods using peripheral light loss as an indicator for successful increased G tolerance.

Total Peripheral Resistance (TPR) was also compared between the two muscle action methods in this study by calculations based on the dependent variables, MAP and CO values. Since the equation used to estimate the value for TPR is based upon MAP and CO measurements (TPR= MAP/CO), it is obvious that there was no statistical significant difference in TPR between pushing and tensing isometric contractions since there was no significant difference between both CO and MAP.

The lack of significant difference between the TPR values for the two contraction methods as demonstrated in this study may be a reflection of an equivalent physiological response between the two muscle contraction methods. While the study retained the null hypothesis, it must be noted that the results fail to demonstrate that either contraction method is superior. The suggestion that the two methods of isometric muscle contractions are equivalent may be significant in itself with regards to performing an effective AGSM. The results indicate that while performing the muscle contraction part of the AGSM, it does not make a difference with regards to blood pressure and cardiac output. Both methods seem to be effective. Future investigators may choose to further explore this.

Conclusion

The two methods of muscle contraction were studied in an effort to determine whether or not one was superior to the other in producing the desired increase in Mean Arterial Pressure (MAP) and Cardiac Output (CO). The results of this study showed no

significant difference between the two methods for either MAP and CO values. The null hypothesis was accepted supporting current literature that suggests no appreciable difference between the two methods exists. The suggestion that the two methods of isometric muscle contractions are equivalent may be significant with regards to performing an effective AGSM. Results indicate that while performing the muscle contraction element of the AGSM, whether the contraction is a pushing or a tensing maneuver, there is no appreciable difference with regards to elevation of blood pressure (MAP) or cardiac output (CO). Both methods appear to be effective in producing the desired physiologic effects. These findings could result in a modification of the present U.S. Air Force AGSM procedure, allowing pilots to use either the tense or the push contractions to increase G tolerance.

Recommendations for Further Research

1. Future repeat testing utilizing a spirometer would more easily eliminate the possibility of subject performing a Valsalva maneuver.
2. Further studies are needed to assess the muscle strategy used for both isometric contraction methods.
3. Utilization of a larger population with an operational experience performing the AGSM is recommended.
4. Use of a more sensitive blood pressure analyzer, possibly invasive, could more accurately identify differences in blood pressure.
5. Performance of the study in a human centrifuge to evaluate both methods of isometric muscle contractions under increased G may yield more valid results.

APPENDIX A

SUBJECT DATA

Appendix A1: Physical Characteristics of Subjects

				Sum of Four (mm) Skinfolds					
Subject	Age	Weight (Kg)	Height (cm)	Abdomen	Illium	Thigh	Triceps	Sum of 4	% Body Fat
1	27	87.0	183.0	25	20	13	7	65	15.1
2	25	72.5	175.0	22	13	8	8	51	11.6
3	22	72.0	173.5	27	28	15	14	84	18.6
4	23	81.0	177.0	6	6	7	5	24	5.2
5	24	72.7	174.0	20	26	11	7	64	15.1
6	24	106.7	192.5	38	51	22	18	129	24.2
7	38	74.5	177.0	26	21	10	15	72	18.6
8	22	106.0	182.0	32	35	25	19	111	23.5
9	22	77.0	179.5	25	18	10	9	62	13.1
10	19	82.0	176.0	27	19	11	13	70	15.4
11	24	60.0	162.5	10	9	6	6	31	6.5
12	18	54.5	169.5	7	7	5	6	25	4.4
Average	24	78.8	176.8	22	21	12	11	66	14.3
SD	5.0	15.6	7.4	9.9	12.8	6.1	5.0	32.0	6.6

Appendix A2: Force Measurements during the Pushing Method

Subject	Angle of Knee	Kilograms	Pounds	Newtons
1	80	75	165	734
2	88	100	220	979
3	89	70	154	685
4	88	100	220	979
5	90	130	287	1277
6	80	120	265	1179
7	90	120	265	1179
8	84	80	176	783
9	82	110	243	1081
10	84	90	198	881
11	90	110	243	1081
12	84	100	220	979
Average	86	100	220	979
SD	4	19	42	187

APPENDIX B

INFORMED CONSENT

Appendix B1: Informed Consent

University of Nevada, Las Vegas
Department of Kinesiology

Informed Consent

Title: Comparative Analysis of Two Methods of Isometric Muscle Contractions During the Anti-G Straining Maneuver.

Purpose:

The purpose of this study is to compare the effectiveness of the Anti-G Straining Maneuver's muscle contractions performed during two different isometric variations.

The standard U. S. Air Force procedure of performing the Anti-G Straining Maneuver, which consists of an isometric contraction or tensing of all the muscles of the lower extremities, will be compared to a technique involving pushing maximally against the floor of the aircraft or as in this study, a footrest platform. It is proposed that this technique of "pushing against the floor" may lead to an increased total peripheral resistance over that technique currently employed, ultimately raising the both Cardiac Output (CO) and Mean Arterial Pressure (MAP). Under most combat and aerobatic maneuvers the pilot is subjected to forces up to nine times his body weight. This increased G load has a profound effect on the blood tissue, causing it to pool into the lower parts of the body. Without any intervention this draining of blood from the brain may cause the pilot to lose consciousness, a state called G-induced Loss of Consciousness or G-LOC. The results of this study may lead to a modification of the present USAF muscle contraction technique.

Explanation of the Tests:

The testing will take place in the Exercise Physiology Laboratory located in the McDermott Center (MPE 326) at the University of Nevada, Las Vegas. This study requires you to commit to a 2-hour testing schedule. You will be asked to perform 2 methods of isometric muscle contraction maneuvers. The first hour will consist of several preliminary tasks, a briefing on the Anti-G Straining Maneuver (isometric muscle tensing contraction) and familiarization session with the testing equipment to be used.

The second hour will consist of actual data collection. Two variations of muscle contractions will be assessed; an isometric tensing contraction of the muscles of the lower extremities (current AGSM technique), and an isometric contraction technique, which consists of forcefully pushing against an immovable foot platform. Both contractions will be performed while strapped into a chair using a five-point harness. The purpose of the harness is to provide a means of resistance while pushing against the footrest platform. Cardiac output will be measured throughout both contraction variations lasting 30 seconds using a non-invasive bioimpedance cardiography analyzer. These measurements are taken from two foil strips surrounding the chest and neck. Blood pressure will also be measured during the 30-second muscle contractions using a sphygmomanometer on the left upper arm.

Risks and Discomforts:

Whenever physically stressing adults, there are possible risks, however, the risks appear to be minimal. You may experience muscle stain and/or muscle soreness. Elevated blood pressure increases the risk of cardiovascular disorders such as heart attack or stroke. However, since subjects will have normal resting blood pressure, and be otherwise healthy, this risk seems minimal. If an injury does occur UNLV will not provide any financial compensation.

Benefits from Testing:

If the proposed technique of isometric contraction while pushing against the floor, which is the same as pushing on the floor of the aircraft cockpit, increases venous peripheral resistance, the expected result would be to improve the overall resistance to acceleration induced loss of consciousness. If this is correct, then the present USAF technique/method might be reevaluated.

Confidentiality:

The data collected during this study is confidential. Only those persons who are directly related to this study (i.e.: researchers, data analysts) will have access to your file. All records will be stored in a locked facility within the Exercise Physiology Laboratory at UNLV. If the results of this study are published, no subject names will be used, instead numbers or codes will be used.

Freedom of Consent:

Your permission to be in this study and to perform these tests is strictly voluntary. You are free to stop the testing at any point, without any penalty.

Inquiries:

Questions regarding this study's significance, purpose, methodology, procedures or risks are encouraged. Your questions and concerns will be addressed to your satisfaction. Inquiries should be directed toward Lance Annicelli or Dr. Lawrence Golding at 895-3766. Please contact the UNLV Office for the Protection of Research Subjects at 895-2794 for additional questions regarding the subject's rights of research.

I have read this form carefully and I am aware of tests/procedures to be performed and the possible risks involved. Any questions have been answered to my satisfaction. I consent to participate in this test. I understand that I have the right to withdraw from this study at any time without prejudice.

Name of Subject	Signature of Subject	Date

Name of Witness	Signature of Witness	Date

Appendix B2: Data Collection Worksheet

COMPARATIVE ANALYSIS OF TWO METHODS OF ISOMETRIC MUSCLE CONTRACTIONS DURING THE ANTI-G STRAINING MANEUVER

Test subject's name: _____

Email address and phone number: _____

Date of birth: _____/_____/_____ Age: _____ Weight: _____kg Height: _____cm

Resting Blood Pressure (must be <140/90 mmHg to continue with this study): _____mmHg

Jackson and Pollock Sum of Four Skinfolds (mm):

1. Abdomen-vertical fold - 1 inch to the right of the umbilicus _____
2. Illium-diagonal fold - just above the crest of the illium on the midaxillary line _____
3. Vertical fold of the thigh - midway between the top of the patella and groin line _____
4. Triceps-vertical fold - back of the upper arm midway between the acromion and olecranon process _____

Total _____

Percent body fat: _____% Angle of knee while seated: _____

Maximum voluntary "Push" contraction: _____kg (collect during orientation session)

Test Protocol

	Isometric **Push** Contraction				Isometric **Tensing** Contraction (AGSM)			
Trial	Blood Pressure (Systolic/Diastolic) mmHg	Cardiac Output L/min	Stroke Volume ml/beat	Heart Rate bpm	Blood Pressure (Systolic/Diastolic) mmHg	Cardiac Output L/min	Stroke Volume ml/beat	Rate bpm
1								
2								
3								

Mean Arterial Pressure (MAP) = Diastolic + 1/3(Systolic – Diastolic)

_____ mmHg	_____ mmHg

Total Peripheral Resistance from MAP/CO

_____ mmHg/L/min	_____ mmHg/L/min

Appendix B3: Human Subjects Approval

UNLV
UNIVERSITY OF NEVADA LAS VEGAS

Biomedical Sciences Institutional Review Board Approval Notice

DATE: November 04, 2002

TO: Lance Annicelli
Lawrence A. Golding Ph.D., Advisor
M/S 3034

FROM: for Dr. Jack Young, Chair JK
UNLV Biomedical Sciences Institutional Review Board

RE: Status of Human Subject Protocol Entitled: ***Comparative Analysis of Two Variations of Isometric Muscle Contractions during the Anti-G Straining Maneuver***

OPRS# 504S0902-486

This memorandum is official notification that the UNLV Biomedical Sciences Institutional Review Board has **approved** the protocol for the project listed above and research on the project may proceed. This approval is effective from the data of this notification and will continue through November 04, 2003, a period of one year from the initial review.

Should the use of human subjects described in this protocol continue beyond a one-year period from the initial review, it will be necessary to request an extension. Should you initiate **ANY** changes to the protocol, it will be necessary to request additional approval for such change(s) in writing through the Office for the Protection of Research Subjects.

If you have questions or require any assistance, please contact the Office for the Protection of Research Subjects at 895-2794.

Cc: OPRS File

Office for the Protection of Research Subjects
4505 Maryland Parkway • Box 451046 • Las Vegas, Nevada 89154-1046
(702) 895-2794 • FAX: (702) 895-0805

APPENDIX C

RAW DATA

Appendix C1: Blood Pressure Measurements

Push Method

Systolic/Diastolic Blood Pressure (mmHg)

Subject	Trial 1	Trial 2	Trial 3	Avg BP
1	152/100	154/104	152/100	153/101
2	152/90	142/96	152/94	149/93
3	128/92	138/96	140/98	135/95
4	166/105	177/102	171/109	171/105
5	140/100	130/110	140/110	137/107
6	160/122	180/140	186/124	175/129
7	152/94	160/95	160/95	156/95
8	150/90	150/90	150/100	150/93
9	160/110	170/120	172/110	167/113
10	162/120	166/121	168/118	165/120
11	163/120	162/112	163/112	163/115
12	170/90	168/95	170/110	169/98
Average				161/107
SD				13/12

Tense Method

Systolic/Diastolic Blood Pressure (mmHg)

Subject	Trial 1	Trial 2	Trial 3	Avg BP
1	132/90	128/92	136/86	132/89
2	158/108	170/106	162/112	163/109
3	144/104	134/100	130/102	136/102
4	160/90	157/91	172/92	162/91
5	138/95	140/95	134/80	137/90
6	156/98	160/108	164/100	160/102
7	165/98	163/98	164/100	164/99
8	170/90	160/100	160/100	163/97
9	165/118	no data	162/115	164/117
10	162112	154/112	144/98	153/107
11	16298	160/100	160102	161/100
12	150/80	160/90	158/98	156/89
Average				154/99
SD				12/9

Appendix C2: Average Change in Blood Pressure Between Subjects

Avg Systolic BP (mmHg)

Subject	Push	Tense	BP Change
1	153	132	21
2	149	163	14
3	135	136	1
4	171	162	9
5	137	137	0
6	175	160	15
7	156	164	8
8	150	163	13
9	167	164	3
10	165	153	12
11	163	161	2
12	169	156	13
Avg	161	154	9
SD	13	12	7

Avg Diastolic BP (mmHg)

Push	Tense	BP Change
101	89	12
93	109	16
95	102	7
105	91	14
107	90	17
129	102	27
95	98.7	3.7
93	97	4
113	117	4
120	107	13
115	100	15
98	89	9
107	99	12
12	9	7

Appendix C3: Cardiac Output Measurements

Push Method

Cardiac Output (L/min)

Subject	Trial 1	Trial 2	Trial 3	Avg CO
1	6.6	6.9	6.9	6.8
2	8.4	7.9	6.9	7.7
3	7.4	7.1	7.3	7.3
4	11.8	11.2	10.8	11.3
5	6.1	7.8	7.3	7.1
6	8.8	8.8	9.1	8.9
7	4.6	5.7	5.4	5.2
8	9.8	6.5	no data	8.2
9	8.6	6.8	6.8	7.4
10	8.9	8.9	8.5	8.8
11	5.6	5.0	6.8	5.8
12	7.1	6.3	7.5	7.0
Average				7.6
SD				1.6

Tense Method

Cardiac Output (L/min)

Subject	Trial 1	Trial 2	Trial 3	Avg CO
1	6.3	6.2	7.7	6.7
2	8.6	9.6	7.9	8.7
3	7.2	6.9	7.0	7.0
4	8.0	9.6	9.6	9.1
5	9.9	8.2	7.6	8.6
6	11.3	11.7	10.2	11.1
7	6.8	6.8	6.4	6.7
8	9.5	11.6	11.5	10.9
9	5.7	no data	5.9	5.8
10	9.9	8.1	8.4	8.8
11	5.0	5.2	4.8	5.0
12	7.5	5.5	5.5	6.2
Avg				7.9
SD				2.0

Appendix C4: Change in Cardiac Output Between Subjects

Average Cardiac Output (L/min)

Subject	Avg Push	Avg Tense	CO Change
1	6.8	6.7	0.1
2	7.7	8.7	1
3	7.3	7.0	0.3
4	11.3	9.1	2.2
5	7.1	8.6	1.5
6	8.9	11.1	2.2
7	5.2	6.7	1.5
8	8.2	10.9	2.7
9	7.4	5.8	1.6
10	8.8	8.8	0
11	5.8	5.0	0.8
12	7.0	6.2	0.8
Avg	7.6	7.9	1.2
SD	1.6	2.0	0.9

Appendix C5: Stroke Volume Measurements

Push Method

Stroke Volume (ml/beat)

Subject	Trial 1	Trial 2	Trial 3	Avg SV
1	77.2	84.5	85.3	82.3
2	90.8	85.3	72	82.7
3	72.2	73.3	74.9	73.5
4	127	119.5	108.4	118.1
5	43.1	61	56	53.4
6	90.7	83.7	89.3	87.9
7	43.8	49.3	49.4	47.5
8	111	70	no data	90.5
9	63.7	60.1	57.2	60.3
10	84.9	90.5	75.3	83.6
11	83.8	83.7	77.9	81.8
12	75.6	68.5	76.5	73.5
Avg				77.9
SD				18.7

Tense Method

Stroke Volume (ml/beat)

Subject	Trial 1	Trial 2	Trial 3	Avg SV
1	81	82	95.1	86.0
2	88.3	113.5	93.8	98.5
3	70.3	65.1	66.1	67.2
4	85.5	110.6	106.1	100.7
5	88.7	82.3	80.5	83.8
6	92.1	109.3	97.1	99.5
7	62.9	62.8	60.2	62.0
8	118	132.5	134.3	128.4
9	49.7	no data	48.2	49.0
10	101	98.5	100.8	100.0
11	73.8	82.1	77.9	77.9
12	72.9	59.3	55.5	62.6
Avg				84.6
SD				22.2

Appendix C6: Change in Stroke Volume Between Subjects

Average Stroke Volume (ml/beat)

Subject	Push	Tense	SV Change
1	82.3	86.0	3.7
2	82.7	98.5	15.8
3	73.5	67.2	6.3
4	118.1	100.7	17.4
5	53.4	83.8	30.4
6	87.9	99.5	11.6
7	47.5	62.0	14.5
8	90.5	128.4	37.9
9	60.3	49.0	11.3
10	83.6	100.0	16.4
11	81.8	77.9	3.9
12	73.5	62.6	10.9
Avg	77.9	84.6	15.0
SD	18.7	22.2	10.2

Appendix C7: Heart Rate and Mean Arterial Pressure Measurements

Push Method

Heart Rate (bpm)

Subject	Trial 1	Trial 2	Trial 3	Avg HR
1	86	82	81	83
2	93	92	96	94
3	102	97	98	99
4	93	93	100	95
5	142	128	130	133
6	97	105	102	101
7	104	115	109	109
8	88	93	no data	91
9	135	113	118	122
10	105	98	113	105
11	67	60	87	71
12	93	92	98	94
Avg				100
SD				16

MAP (mmHg)

MAP
118
112
108
127
117
144
115
112
131
135
131
122
123
11

Tense Method

Heart Rate (bpm)

Subject	Trial 1	Trial 2	Trial 3	Avg HR
1	78	76	81	78
2	97	85	84	89
3	103	106	106	105
4	94	87	90	90
5	112	99	94	102
6	123	107	106	112
7	108	108	107	108
8	80	88	85	84
9	116	no data	122	119
10	98	83	83	88
11	67	63	61	64
12	103	92	100	98
Avg				95
SD				16

MAP (mmHg)

MAP
104
127
113
115
106
121
120
119
134
122
120
111
118
8

Appendix C8: Change in Heart Rate and Mean Arterial Pressure Between Subjects

Avg Heart Rate (bpm)

Subject	Push	Tense	HR Change
1	83	78	5
2	94	89	5
3	99	105	6
4	95	90	5
5	133	102	31
6	101	112	11
7	109	108	1
8	91	84	7
9	122	119	3
10	105	88	17
11	71	64	7
12	94	98	4
Avg	100	95	9
SD	16.5	15.6	8.3

Avg Mean Arterial Pressure (mmHg)

Subject	Push	Tense	MAP Change
1	118	104	14
2	112	127	15
3	108	113	5
4	127	115	12
5	117	106	11
6	144	121	23
7	115	120	5
8	112	119	7
9	131	134	3
10	135	122	13
11	131	120	11
12	122	111	11
Avg	123	118	11
SD	11.0	8.5	5.4

Appendix C9: Total Peripheral Resistance Measurements

Push Method

Subject	Avg (mmHg/L/min)	Avg (Dynes/sec/cm-5)
1	17.4	1393
2	14.5	1160
3	14.8	1184
4	11.2	896
5	16.4	1312
6	16.2	1296
7	22.1	1768
8	13.7	1099
9	17.7	1416
10	15.3	1227
11	22.6	1808
12	17.4	1392
Average	16.6	1329
SD	3.2	259

Tense Method

Subject	Avg (mmHg/L/min)	Avg Dynes/sec/cm-5
1	15.4	1236
2	14.6	1168
3	16.2	1296
4	12.6	1008
5	12.3	984
6	10.9	872
7	18.0	1440
8	11.0	876
9	23.1	1848
10	14.0	1120
11	24.0	1920
12	17.9	1440
Average	15.8	1267
SD	4.3	346

Appendix C10: Change in Total Peripheral Resistance Between Subjects

Average Total Peripheral Resistance (mmHg/L/min)

Subject	Push	Tense	TPR Change
1	17.4	15.4	2.0
2	14.5	14.6	0.1
3	14.8	16.2	1.4
4	11.2	12.6	1.4
5	16.4	12.3	4.1
6	16.2	10.9	5.3
7	22.1	18.0	4.1
8	13.7	11.0	2.7
9	17.7	23.1	5.4
10	15.3	14.0	1.3
11	22.6	24.0	1.4
12	17.4	17.9	0.5
Avg	16.6	15.8	2.5
SD	3.2	4.3	1.8

Appendix C11: Predicted Cardiac Output at Rest

Subject	Weight (lbs)	Height (inch)	Body Surface Area	Predicted CO (BSA x 2.8)
1	192	72.0	2.06	5.77
2	160	68.9	1.86	5.21
3	159	68.3	1.84	5.15
4	179	70.0	1.96	5.49
5	160	68.5	1.86	5.21
6	235	75.8	2.36	6.61
7	164	69.7	1.91	5.35
8	234	71.7	2.26	6.33
9	170	70.7	1.95	5.46
10	181	69.3	1.96	5.49
11	132	64.0	1.61	4.51
12	120	66.7	1.61	4.51
Avg	173.8	69.6	1.9	5.4
SD	34.6	2.9	0.2	0.6

Predicted Cardiac Output calculated from Body Surface Area (BSA) (Dubois, B.S., & Dubois, E.F., 1916).

Appendix C12: Absolute Difference Between Push and Tense Contractions

Subject	CO (L/min)		SV (ml/beat)		HR (bpm)		MAP (mmHg)		TPR (mmHg/L/min)	
1	0.1	P	3.7	T	5	P	14	P	2	P
2	1	T	15.8	T	5	P	15	T	0.1	T
3	0.3	P	6.3	P	6	T	5	T	1.4	T
4	2.2	P	17.4	P	5	P	12	P	1.4	T
5	1.5	T	30.4	T	31	P	11	P	4.1	P
6	2.2	T	11.6	T	11	T	23	P	5.3	P
7	1.5	T	14.5	T	1	P	5	T	4.1	P
8	2.7	T	37.9	T	7	P	7	T	2.7	P
9	1.6	P	11.3	P	3	P	3	T	5.4	T
10	0		16.4	T	17	P	13	P	1.3	P
11	0.8	P	3.9	P	7	P	11	P	1.4	T
12	0.8	P	10.9	P	4	T	11	P	0.5	T

Data indicates absolute difference between contraction methods. P indicates difference favorable for Push and T indicates favorable for Tense.

Appendix C13: EMG Muscle Activation Raw Data

Subject	Muscle	Trial 1 Push 1	Trial 1 Tense 1	Trial 2 Push 2	Trial 2 Tense 2	Trial 3 Push 3	Trial 3 Tense 3	Trial 4 Push 4	Trial 4 Tense 4	Avg Push	Avg Tense
7	Gluteus Maximus	13.8	13.2	15.1	14.6	19	18.4	**16**	10.2	**16.0**	14.1
	Rectus Femoris	**30.3**	20.2	**41.8**	29.5	**52**	17.9	**50.2**	23.9	**43.6**	22.9
	Vastus Medialis	**80.4**	15.5	**99.2**	43.6	**127**	15.3	**119.5**	31.9	**106.5**	26.6
	Vastus Lateralis	**46.9**	11	**58.1**	23.5	**79.2**	10	**67.9**	20	**63.0**	16.1
	Biceps Femoris	**25.8**	10.6	**34.6**	22	**42.5**	12.2	**35**	18.1	**34.5**	15.7
	Gastrocnemius Medial	18.9	**21.8**	18.9	19.2	**23.4**	18.8	19.6	18.9	**20.2**	19.7

Subject	Muscle	Push 1	Tense 1	Push 2	Tense 2	Push 3	Tense 3	Avg Push	Avg Tense
4	Gluteus Maximus	12.8	**26.7**	19.3	**27.8**	21	**30.8**	17.7	**28.4**
	Rectus Femoris	**60.1**	57.1	60.5	**73.6**	60.9	**75.7**	60.5	**68.8**
	Vastus Medialis	**103.7**	26.3	**99.5**	34.5	**111**	30.2	**104.7**	30.3
	Vastus Lateralis	**111.7**	36.8	**116.2**	49.8	**139.7**	44.2	**122.5**	43.6
	Biceps Femoris	11	**33.6**	28.5	**55.1**	33.2	**51.8**	24.2	**46.8**
	Gastrocnemius Medial	18.8	**50.1**	33.7	**63.8**	26.2	**49.5**	26.2	**54.5**

Subject	Muscle	Push 1	Tense 1	Push 2	Tense 2	Push 3	Tense 3	Avg Push	Avg Tense
5	Gluteus Maximus	**27**	7	**19.4**	3.2	**19.9**	3.7	**22.1**	4.6
	Rectus Femoris	**75.8**	17.8	**59.1**	25.8	**65.4**	19	**66.8**	20.9
	Vastus Medialis	**90.1**	11.3	**73.8**	14.1	**76.9**	9.4	**80.3**	11.6
	Vastus Lateralis	**148.3**	21	**117.4**	25.9	**127.1**	16.7	**130.9**	21.2
	Biceps Femoris	17.9	**37.6**	15.9	**18.4**	18	**27.5**	17.3	**27.8**
	Gastrocnemius Medial	18.7	**46.4**	18.7	**57.9**	18.8	**24.8**	18.7	**43.0**

EMG data are displayed as average microvolts. Subject number corresponds to original test subject data identification. Numbers in bold represent larger number of muscle activity detected between the two methods of isometric contractions.

Appendix C14: EMG Muscle Activation Raw Data

Muscle	Subject 7 Avg Push	Subject 4 Avg Push	Subject 5 Avg Push	Average	SD
Gluteus Maximus	**16**	17.7	**22.1**	18.6	3.148
Rectus Femoris	**43.6**	60.5	**66.8**	57.0	11.997
Vastus Medialis	**106.5**	**104.7**	**80.3**	97.2	14.635
Vastus Lateralis	**63**	**122.5**	**130.9**	105.5	37.016
Biceps Femoris	**34.5**	24.2	17.3	25.3	8.656
Gastrocnemius Medial	**20.2**	26.2	18.7	21.7	3.969
Muscle	Avg Tense	Avg Tense	Avg Tense		
Gluteus Maximus	14.1	**28.4**	4.6	15.7	11.980
Rectus Femoris	22.9	**68.8**	20.9	37.5	27.096
Vastus Medialis	26.6	30.3	11.6	22.8	9.903
Vastus Lateralis	18.2	43.6	21.2	27.7	13.880
Biceps Femoris	15.7	**46.8**	**27.8**	30.1	15.677
Gastrocnemius Medial	19.7	**54.5**	**43**	39.1	17.730

EMG data are displayed as average microvolts. Numbers in bold represent larger number of muscle activity detected between the two methods of isometric contractions.

	Subject 7		Subject 4		Subject 5	
Muscle	Push	Tense	Push	Tense	Push	Tense
Gluteus Maximus	15%	53%	14%	41%	17%	11%
Rectus Femoris	41%	86%	49%	100%	51%	49%
Vastus Medialis	100%	100%	85%	44%	61%	27%
Vastus Lateralis	59%	68%	100%	63%	100%	49%
Biceps Femoris	32%	59%	20%	68%	13%	65%
Gastrocnemius Medial	19%	74%	21%	79%	14%	100%

EMG data normalized to largest muscle activity displayed in microvolts. 100 % indicates maximum muscle activity detected under each method of contraction.

APPENDIX D

CONVERSION CHARTS

Appendix D1: Jackson and Pollock Sum of 4 Skinfolds Conversion Chart (Golding, 2000)

	Age to Last Year								
Sum of 4 Skinfolds	18-22	23-27	28-32	33-37	38-42	43-47	48-52	53-57	58
13-17	1.7	2.5	3.3	4.1	4.9	5.6	6.4	7.2	8.0
18-22	3.1	3.9	4.6	5.4	6.2	7.0	7.8	8.6	9.4
23-27	4.4	5.2	6.0	6.8	7.6	8.4	9.2	10.0	10.7
28-32	5.7	6.5	7.3	8.1	8.9	9.7	10.5	11.3	12.1
33-37	7.0	7.8	8.6	9.4	10.2	11.0	11.8	12.6	13.4
38-42	8.3	9.1	9.9	10.7	11.5	12.3	13.1	13.9	14.6
43-47	9.6	10.3	11.1	11.9	12.7	13.5	14.3	15.1	15.9
48-52	10.8	11.6	12.4	13.2	13.9	14.7	15.5	16.3	17.1
53-57	12.0	12.8	13.6	14.4	15.1	15.9	16.7	17.5	18.3
58-62	13.1	13.9	14.7	15.5	16.3	17.1	17.9	18.7	19.5
63-67	14.3	15.1	15.9	16.7	17.5	18.2	19.0	19.8	20.6
68-72	15.4	16.2	17.0	17.8	18.6	19.4	20.2	21.0	21.8
73-77	16.5	17.3	18.1	18.9	19.7	20.5	21.3	22.1	22.8
78-82	17.6	18.4	19.2	20.0	10.7	21.5	22.3	23.1	23.9
83-87	18.6	19.4	20.2	21.0	21.8	22.6	23.4	24.2	25.0
88-92	19.6	10.4	21.2	22.0	22.8	23.6	24.4	25.2	26.0
93-97	20.6	21.4	22.2	23.0	23.8	24.6	25.4	26.2	27.0
98-102	21.6	22.4	23.2	24.0	24.8	25.6	26.4	27.1	27.9
103-107	22.5	23.3	24.1	24.9	25.7	26.5	27.3	28.1	28.9
108-112	23.5	24.2	25.0	25.8	26.6	27.4	28.2	29.0	29.8
113-117	24.3	25.1	25.9	26.7	27.5	28.3	29.1	29.9	30.7
118-122	25.2	26.0	26.8	27.6	28.4	29.2	30.0	30.8	31.6
123-127	26.0	26.8	27.6	28.4	29.2	30.0	30.8	31.6	32.4
128-132	26.9	27.7	28.4	29.2	30.0	30.8	31.6	32.4	33.2
133-137	27.7	28.4	29.2	30.0	30.8	31.6	32.4	33.2	34.0
138-142	28.4	29.2	30.0	30.8	31.6	32.4	33.2	34.0	34.8
143-147	29.2	29.9	30.7	31.5	32.3	33.1	33.9	34.7	35.5
148-152	29.9	30.7	31.5	32.2	33.0	33.8	34.6	35.4	36.2
153-157	30.6	31.3	32.1	32.9	33.7	34.5	65.3	36.1	36.9
158-162	31.2	32.0	32.8	33.6	34.4	35.2	36.0	36.8	37.6
163-167	31.8	32.6	33.4	34.2	35.0	35.8	36.6	37.4	38.2
168-172	32.5	33.3	34.0	34.8	35.6	36.4	37.2	38.0	38.8
173-177	33.0	33.8	34.6	35.4	36.2	37.0	37.8	38.6	39.4
178-182	33.6	34.4	35.2	36.0	36.8	37.6	38.4	39.2	39.9
183-187	34.1	34.9	35.7	36.5	37.3	38.1	38.9	39.7	40.5

Appendix D2: Body Surface Area in Square Meters Conversion Chart (DuBois, B.S., & Dubois, E.F., 1916)

Weight in Pounds \ Height in inches	60	61	62	63	64	65	66	67	68
80	1.25	1.26	1.28	1.30	1.32	1.33	1.35	1.36	1.37
85	1.28	1.30	1.32	1.33	1.35	1.37	1.39	1.40	1.41
90	1.31	1.33	1.35	1.36	1.38	1.40	1.42	1.43	1.44
95	1.35	1.36	1.38	1.40	1.42	1.43	1.45	1.46	1.48
100	1.36	1.39	1.41	1.43	1.45	1.46	1.47	1.49	1.51
105	1.41	1.42	1.44	1.46	1.48	1.49	1.50	1.52	1.54
110	1.43	1.45	1.47	1.48	1.50	1.52	1.53	1.55	1.57
115	1.46	1.48	1.50	1.51	1.53	1.55	1.56	1.58	1.60
120	1.49	1.51	1.53	1.54	1.56	1.58	1.59	1.61	1.63
125	1.52	1.54	1.56	1.57	1.59	1.61	1.62	1.64	1.66
130	1.54	1.56	1.58	1.59	1.61	1.63	1.65	1.67	1.68
135	1.57	1.59	1.61	1.62	1.64	1.66	1.68	1.70	1.71
140	1.59	1,61	1.63	1.64	1.66	1.68	1.70	1.72	1,74
145	1.62	1.64	1.66	1.68	1.69	1.71	1.73	1.75	1.77
150	1.64	1.66	1.68	1.69	1.71	1.73	1.75	1.77	1.79
155	1.66	1.68	1.70	1.72	1.74	1.76	1.78	1.80	1.82
160	1.68	1.70	1.72	1.74	1.76	1.78	1.80	1.82	1.84
165	1.71	1.73	1.75	1.77	1.79	1.81	1.83	1.85	1.87
170	1.73	1.75	1.77	1.79	1.81	1.83	1.85	1.87	1.89
175	1.75	1.77	1.79	1.82	1.84	1.86	1.88	1.90	1.92
180	1.77	1.79	1.81	1.84	1.86	1.88	1.90	1.92	1.94
185	1.79	1.81	1.83	1.86	1.88	1.90	1.92	1.94	1.96
190	1.81	1.83	1.85	1.88	1.90	1.92	1.94	1.96	1.98
195	1.83	1.85	1.87	1.90	1.92	1.94	1.96	1.98	2.01
200	1.85	1.87	1.89	1.92	1.94	1.96	1.98	2.00	2.03
205	1.87	1.89	1.91	1.94	1.96	1.98	2.00	2.02	2.05
210	1.89	1.91	1.93	1.96	1.98	2.00	2.02	2.04	2.07
215	1.91	1.93	1.95	1.97	1.99	2.02	2.04	2.06	2.09
220	1.93	1.95	1.97	1.99	2.02	2.04	2.06	2.08	2.11
225	1.95	1.97	1.99	2.01	2.04	2.06	2.08	2.10	2.13
230	1.96	1.99	2.01	2.03	2.05	2.08	2.10	2.12	2.15
235	1.98	2.01	2.03	2.05	2.07	2.10	2.12	2.14	2.17
240	2.00	2.03	2.05	2.07	2.09	2.12	2.15	2.17	2.19
245	2.02	2.05	2.07	2.09	2.11	2.14	2.17	2.19	2.21
250	2.03	2.06	2.08	2.11	2.13	2.16	2.19	2.21	2.24
255	2.05	2.08	2.10	2.13	2.15	2.18	2.21	2.23	2.26
260	2.06	2.09	2.11	2.14	2.16	2.19	2.22	2.24	2.27
265	2.08	2.11	2.13	2.16	2.18	2.21	2.24	2.26	2.29
270	2.10	2.13	2.15	2.17	2.19	2.22	2.25	2.27	2.30
275	2.12	2.15	2.17	2.19	2.21	2.24	2.27	2.29	2.32
280	2.13	2.16	2.18	2.20	2.22	2.25	2.28	2.30	2.33
285	2.15	2.18	2.20	2.22	2.24	2.27	2.30	2.32	2.36
290	2.16	2.19	2.21	2.24	2.26	2.29	2.31	2.33	2.36

Weight in Pounds	Height in inches 69	70	71	72	73	75	75	76	77
80	1.38	1.40	1.42	1.43	1.44	1.45	1.47	1.48	1.49
85	1.42	1.44	1.46	1.47	1.48	1.49	1.51	1.52	1.53
90	1.45	1.47	1.49	1.50	1.52	1.53	1.55	1.56	1.57
95	1.49	1.51	1.53	1.54	1.56	1.57	1.59	1.60	1.61
100	1.53	1.54	1.55	1.57	1.59	1.61	1.62	1.63	1.65
105	1.56	1.57	1.59	1.61	1.62	1.64	1.66	1.67	1.69
110	1.59	1.60	1.62	1.64	1.65	1.67	1.69	1.70	1.72
115	1.62	1.63	1.65	1.67	1.68	1.70	1.72	1.73	1.75
120	1.65	1.66	1.68	1.70	1.71	1.73	1.75	1.76	1.78
125	1.68	1.69	1.71	1.73	1.74	1.76	1.78	1.79	1.81
130	1.70	1.72	1.74	1.76	1.77	1.79	1.81	1.82	1.84
135	1.73	1.75	1.77	1.79	1.80	1.82	1.84	1.86	1.87
140	1.76	1.78	1.80	1.82	1.83	1.85	1.87	1.88	1.90
145	1.79	1.81	1.83	1.85	1.86	1.88	1.90	1.91	1.93
150	1.81	1.83	1.85	1.87	1.89	1.91	1.93	1.95	1.97
155	1.84	1.85	1.88	1.90	1.92	1.94	1.96	1.98	2.00
160	1.86	1.88	1.90	1.92	1.94	1.96	1.98	2.00	2.02
165	1.89	1.91	1.93	1.95	1.97	1.99	2.01	2.03	2.05
170	1.91	1.93	1.95	1.97	1.99	2.01	2.03	2.05	2.07
175	1.94	1.96	1.98	2.00	2.02	2.04	2.06	2.08	2.10
180	1.96	1.98	2.00	2.02	2.04	2.06	2.08	2.10	2.12
185	1.98	2.00	2.02	2.04	2.06	2.08	2.10	2.12	2.14
190	2.00	2.02	2.04	2.06	2.08	2.10	2.12	2.14	2.16
195	2.03	2.05	2.07	2.09	2.11	2.13	2.15	2.17	2.19
200	2.05	2.07	2.09	2.11	2.13	2.15	2.17	2.19	2.21
205	2.07	2.09	2.11	2.13	2.16	2.18	2.20	2.22	2.24
210	2.09	2.11	2.14	2.16	2.18	2.20	2.22	2.25	2.27
215	2.11	2.13	2.16	2.18	2.20	2.22	2.25	2.27	2.30
220	2.13	2.15	2.18	2.20	2.22	2.24	2.27	2.30	2.32
225	2.15	2.17	2.20	2.22	2.24	2.26	2.29	2.32	2.34
230	2.17	2.19	2.22	2.24	2.26	2.28	2.31	2.34	2.36
235	2.19	2.21	2.24	2.26	2.28	2.30	2.33	2.36	2.38
240	2.21	2.24	2.26	2.28	2.30	2.32	2.35	2.38	2.40
245	2.23	2.26	2.28	2.30	2.32	2.34	2.37	2.40	2.42
250	2.26	2.29	2.31	2.33	2.35	2.37	2.39	2.41	2.43
255	2.28	2.31	2.33	2.35	2.37	2.39	2.41	2.43	2.45
260	2.29	2.32	2.34	2.36	2.39	2.41	2.43	2.45	2.47
265	2.31	2.34	2.36	2.38	2.41	2.43	2.45	2.47	2.49
270	2.32	2.35	2.38	2.40	2.42	2.44	2.47	2.50	2.52
275	2.34	2.37	2.40	2.42	2.44	2.46	2.49	2.52	2.54
280	2.35	2.38	2.41	2.43	2.46	2.48	2.51	2.54	2.56
285	2.37	2.40	2.43	2.45	2.48	2.50	2.53	2.56	2.58
290	2.38	2.42	2.45	2.47	2.50	2.52	2.55	2.58	2.60
295	2.40	2.44	2.47	2.49	2.52	2.54	2.57	2.60	2.62

APPENDIX E

STATISTICAL TREATMENT

Appendix E1: Statistical Treatment

t-Test: Paired Two Sample for Means
MAP

	Push	Tense
Mean	122.6667	117.6667
Variance	120.0606	72.0606
Observations	12.0000	12.0000
Pearson Correlation	0.3320	
Hypothesized Mean Difference	0.0000	
df	11.0000	
t Stat	1.5170	
P(T<=t) one-tail	0.0787	
t Critical one-tail	1.7959	
P(T<=t) two-tail	0.1575	
t Critical two-tail	2.2010	

t-Test: Paired Two Sample for Means
CO

	Push	Tense
Mean	7.6250	7.8833
Variance	2.5057	3.8197
Observations	12.0000	12.0000
Pearson Correlation	0.6460	
Hypothesized Mean Difference	0.0000	
df	11.0000	
t Stat	-0.5865	
P(T<=t) one-tail	0.2847	
t Critical one-tail	1.7959	
P(T<=t) two-tail	0.5694	
t Critical two-tail	2.2010	

t-Test: Paired Two Sample for Means
TPR

	Push	Tense
Mean	1329.2500	1267.3333
Variance	67232.0227	119374.0606
Observations	12.0000	12.0000
Pearson Correlation	0.7059	
Hypothesized Mean Difference	0.0000	
df	11.0000	
t Stat	0.8747	
P(T<=t) one-tail	0.2002	
t Critical one-tail	1.7959	
P(T<=t) two-tail	0.4004	
t Critical two-tail	2.2010	

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